1025-95 Technetium 99-m Sestamibi Imaging in the Patient With Cocaine Associated Chest Pain; Alternative to Routine CCU Admissions

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Patients (pts) presenting to the Emergency Department (ED) with chest pain (CP) after cocaine use frequently have ECG abnormalities making it difficult to exclude myocardial ischemia. Many of these pts are admitted and subsequently rule out for MI. Technetium-99m Sestamibi imaging (MIBI) has demonstrated a high sensitivity and specificity in the evaluation of CP to exclude ischemia. Methods: Between January 1994 and July 1995, 103 consecutive pts (mean age 35 ± 7, range 23–54) with cocaine associated CP were evaluated in the ED. Gated MIBI perfusion imaging was performed in 91. Low risk pts were promptly injected with MIBI in the ED and imaged 60–90 minutes later.

Results: Three pts had ECG findings in the ED consistent with a MI and were treated with thrombolytic therapy. Three left AMA. Six were admitted to the CCU without a MIBI and ruled out for a MI. Of the 91 pts who underwent MIBI, 88 (96%) had normal perfusion and normal systolic wall motion. Three pts (4%) had abnormal myocardial perfusion images: 1 had a MI and 2 had ischemic ECG changes with CP but subsequently had a normal catheterization. A total of 25 pts were admitted to the CCU to rule out; none of the patients with a normal MIBI had enzymatic or ECG evidence of a MI.

Conclusion: Myocardial infarction is infrequent in patients presenting with cocaine associated CP (4% in this study). A normal myocardial perfusion study in the ED can identify these pts at low risk for an acute coronary syndrome and may offer a safe alternative to a CCU admission.

1025-96 Effect of Cold Pressor Testing and Iv L-Arginine on Coronary Vasomotion in Patients With Syndrome X

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Previous studies have suggested an impairment in endothelium-independent coronary vasodilation in patients (pts) with Syndrome X. However, coronary vasomotor response to cold (endothelial function) and to intravenous L-arginine, the precursor of EDRF-NO, might also be abnormal in these pts. Therefore, myocardial blood flow (MBF) was quantified under baseline conditions with N-13 ammonia/PET (2 compartment model) at rest, during cold pressor testing (MBF-CPT) and during IV dipyridamole in 10 females (54 ± 11 years) with Syndrome X (pos. treadmill test; neg. hyperventilation test; normal coronary angiography) and in 10 healthy volunteers (n = 10; 45 ± 12 years). In patients, MBF-rest and MBF-CPT were also measured during the IV infusion of L-arginine (6.7 mg/min over 45 minutes). The rate pressure product (RPP) was unaltered by L-arginine at rest (7892 ± 850 vs 8276 ± 1400) and during CPT (11021 ± 2160 vs 11553 ± 2210). Similarly, L-arginine did not affect MBF at rest (0.85 ± 0.16 vs 0.90 ± 0.14 ml/g/min) or during CPT (0.94 ± 0.10 vs 1.04 ± 0.18 ml/g/min). Moreover, CPT increased MBF to a similar degree in controls (23%), patients under baseline conditions (16%) and patients during L-arginine (18%), respectively (p = NS). In contrast, the hyperemic response to IV dipyridamole was blunted (1.72 ± 0.57 vs 2.30 ± 0.32 ml/g/min; $p < 0.05$) resulting in a significant reduction in the myocardial flow reserve in patients relative to controls (1.89 ± 0.62 vs. 3.45 ± 1.03; $p < 0.01$). Thus, coronary vasomotion in response to cold is normal and is unaltered by IV L-arginine suggesting preserved endothelial function in patients with Syndrome X. However, the attenuation in coronary vasodilatory capacity and flow reserve suggests a dysfunction in endothelial independent coronary smooth muscle cell relaxation in these patients.

1025-97 Value of Exercise and Adenosine Myocardial Perfusion Tomography in Patients With Atrial Fibrillation

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Patients (pts) with atrial fibrillation often reach their predicted peak heart rate (PHR) very early during exercise testing. This may be secondary to changes in the conduction system and may be out of proportion to the metabolic demands elicited by exercise. There are no data on the value of stress perfusion tomography for the diagnosis of coronary artery disease (CAD) in those pts. Accordingly, we studied 175 consecutive pts with atrial fibrillation who had exercise or adenosine perfusion tomography in our laboratory. Exercise was used in 100 pts (80 males; mean age 66 ± 10 years) and adenosine in 75 pts (43 males; mean age 72 ± 8 years). The mean exercise duration was 5 ± 2 min and PHR was 157 ± 25 beats/min, with 89% of pts reaching and 52% exceeding PHR. In the adenosine group, maximal heart rate was 96 ± 24 beats/min ($p < 0.01$ vs exercise). Coronary angiography was done within one month of the nuclear study in 82 pts (41 each in the exercise and the adenosine groups). Diameter stenosis (> 50%) was considered significant. The sensitivities and specificities for the overall diagnosis of CAD were 87% an 73% for adenosine and 81% and 67% for exercise tomography, respectively (p = ns). However, the sensitivities and specificities for the diagnosis of left anterior descending artery stenoses were 83% and 89% for adenosine, but only 41% and 75% for exercise. The sensitivities and specificities were similar during either stress for right coronary or circumflex artery stenoses. Thus, in pts with atrial fibrillation, exercise and adenosine tomography have similar sensitivity and specificity for the diagnosis of CAD. During exercise, the premature achievement of PHR lowers the sensitivity for diagnosing left anterior descending artery stenoses, when compared to adenosine tomography.

1026 Coronary Vascular Physiology/Clinical Studies III

Wednesday, March 27, 1996, Noon–2:00 p.m.
Orange County Convention Center, Hall E
Presentation Hour: 1:00 p.m.–2:00 p.m.

1026-15 Endothelial Dysfunction of the Coronary Microcirculation Is Predicted by Epicardial Vessel Constriction in Hypertension

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Hypertension is associated with vasoconstriction of the epicardial vessels in response to the endothelium dependent agent acetylcholine (ACh). The purpose of this study was to determine if endothelial dysfunction of the epicardial vessels in hypertension predicts dysfunction of the microcirculation via an endothelium dependent mechanism. Measurements of coronary epicardial (EP) and microcirculation (MC) vasodilation (mean ± S.E.) were performed during graded intracoronary infusion of ACh. Thirty-one normotensive nondiabetic pts. with normal coronary arteries were the control gp. These demonstrated vasodilation of the EP vessels (3.2 ± 2% increase in diameter) and of the MC (212 ± 22% increase in coronary blood flow (CBF)) at peak effect of ACh. Twenty-eight pts. had HTN without LVH. These showed initial vasodilation, then constriction of the EP (0.5 ± 2.9% increase in the coronary diameter, p = NS) and lesser vasodilation of the MC (193 ± 28% increase in CBF, p = NS) at peak effect of ACh when compared with the control gp. Thirty-nine pts. had HTN with LVH. These showed increasing constriction of the EP (−5 ± 2% increase in the coronary diameter, p = 0.004) and even greater blunting of vasodilation of the MC (95 ± 11% increase in CBF, $p < 0.001$) at peak effect of ACh when compared with the control gp.

Conclusion: Increasing epicardial coronary artery vasoconstriction predicts increasing endothelial dysfunction of the microcirculation with greater clinical severity of hypertension.

1026-16 Influence of Pain on Heart Rate Variability During Holter Monitoring

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The present study was undertaken to evaluate the impact of pain on sympathovagal balance. Accordingly, we evaluated heart rate variability (HRV) during Holter monitoring (HM) in 59 subjects selected as follows: 13 pts with stable coronary artery disease (CAD) and anginal pain during HM (G1); 14 pts with CAD as well, but with totally silent myocardial ischemia (G2); 14 pts with chest pain of non cardiac origin (gastro-esophageal disorders or musculoskeletal pain syndromes: N1) and 18 asymptomatic normal subjects (N2). The 4 groups were age and sex matched. All pts underwent a 24-hr HM off drugs, and accurate diaries were obtained. Tapes were then analysed to assess transient myocardial ischemia and time domain measures of HRV (SDNN, SDANN, rMSSD, pNN50). No difference in the severity of transient ischemia, as assessed by HM, was found in CAD pts: ischemic episodes

	SDNN	SDANN	rMSSD	pNN50
G1	109 ± 23 ms*	99 ± 24 ms*	24 ± 7 ms**	4.4 ± 3.9%**
G2	151 ± 26 ms	134 ± 22 ms	45 ± 19 ms	18.5 ± 12.4%
N1	107 ± 15 ms*	99 ± 17 ms*	25 ± 8 ms**	5.6 ± 5.6%**
N2	153 ± 25 ms	144 ± 27 ms	37 ± 15 ms	13.8 ± 13.1%

*$p < 0.001$, **$p < 0.03$